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Editorial

Botulism: a potentially common problem

Botulism is rare in almost all developed countries and has been for many years. Why then should we fear that it might become a problem? The answer is that its incidence is closely linked to methods of food preparation, and these are continually changing. New methods, such as vacuum preparation of food, provide ideal conditions for the production of botulinum toxin.

Botulism is unknown after eating fresh food. Fermentation, pickling, canning, smoking, and similar processes that require anaerobic conditions enable *Clostridium botulinum* to produce its toxin. Botulism may follow unless the food is adequately heated before consumption. Although one of the most potent toxins known, botulinum toxin is easily inactivated by, for instance, heating to 80°C for only five minutes.

Botulinum toxin prevents the release of acetylcholine from the presynaptic membrane of synapses and the neuromuscular junction.[1] Seven toxins (A–G) are recognised by their serological differences, but only toxins A, B, and E cause human botulism. The toxins have no primary effect on nerve conduction or muscle function,[2] but their action at the motor end plate effectively denervates the muscle and this has very important consequences. Histochemical changes in the muscle fibres develop and the difference between type I and type II fibres becomes less distinct.[3] Sprouting of new nerve terminals and the formation of new motor end plates are seen in affected muscles.[4,5] The toxin appears to be irreversibly bound to the nerve terminals and antitoxin is of no value once this binding has taken place. Recovery of function therefore depends on the formation of new motor end plates from the sprouting nerve terminals.

The clinical presentation of botulism reflects the effects of the toxin on the neuromuscular junction and autonomic synapses. Symptoms due to skeletal muscle weakness and autonomic dysfunction appear after a latent period of usually 12–72 hours from the ingestion of the contaminated food. The muscles supplied by the cranial nerves weaken first, but respiratory muscle weakness usually follows. Nausea, vomiting, dry mouth, constipation, and urinary retention are common. The patient remains afebrile and conscious and sensory signs do not develop. The differential diagnosis includes the Guillain-Barré syndrome, acute poliomyelitis, myasthenia gravis, the Lambert-Eaton syndrome, tick paralysis, and chemical poisoning by, for instance, atropine and organophosphorus compounds. The diagnosis is confirmed by observing the effects of injection of the patient's serum into mice and by isolation of botulinum toxin from food residues.

Respiratory muscle weakness often progresses rapidly and its severity is easily underestimated. Respiratory arrest still occurs commonly before mechanical support and has been started. Blood gas alterations appear late, as in many other neuromuscular disorders, and should not be relied on. Frequent careful clinical assessment and regular measurements of vital capacity are essential. These may be supplemented by maximal inspiratory and expiratory pressure estimations if these are practicable. A rapid fall in vital capacity, a vital capacity of only a third of the predicted value, or a value of around one litre should all suggest the need for ventilatory support.[6]

Ventilatory support may also be needed because of failure to clear secretions and the risk of inhalation even if the vital capacity is only mildly abnormal. Aspiration pneumonia is common and it is important that the method of ventilatory support should protect the airway. Modern methods of ventilatory assistance that are valuable for domiciliary use, such as negative pressure and nasal intermittent positive pressure ventilation, are not suitable. Positive pressure ventilation via an endotracheal tube is required. A tracheostomy is usually needed, except in mild cases, since recovery of muscle function is usually slow. Ventilatory support is often needed for several weeks.[6] Infections due to type A toxin are particularly severe and usually require a long period of ventilatory support and a long hospital stay.[7,8] A short latent period before symptoms appear is also correlated with a more severe illness.[9] Mortality during the period of ventilation used to be high, but with modern intensive care methods death should be unusual. Most deaths are now caused by failure to recognise the severity and rapidity of progression of respiratory muscle weakness in its early stages.

All the studies of the late results of botulism emphasise the persistence of the symptoms of muscle weakness. Fatigue, breathlessness on exertion, and diplopia are common.[6,10] Physiological studies have not, however, shown any appreciable abnormalities of respiratory muscle function. Lung volumes, FEV_1/

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FVC, maximal inspiratory and expiratory pressures, the ventilatory response to exercise, and arterial blood gases have all been normal.[10] These findings may be misleading as they do not directly reflect the state of neuromuscular transmission, inadequacy of which is the primary defect in botulism. The hallmark of this is muscle fatiguability. This can be demonstrated in the acute phase by a positive edrophonium (Tensilon) test.[11] Studies of the endurance of the respiratory and other muscles in the convalescent stage are needed to show whether the late symptoms of botulism reflect incomplete recovery from the direct effects of the toxin.

Botulism is a severe and frightening illness for the patient. Depression and loss of confidence are common and may contribute to the slow return to a normal life.[12] It is important that the patient has realistic expectations about his abilities at each stage of his illness. Respiratory rehabilitation programmes that include progressive exercise training regimens may prove valuable during the recovery phase. Botulism, though rare at present, may well become more common and could be the stimulus for the development of rehabilitation techniques and facilities that will be useful in many other respiratory disorders.

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